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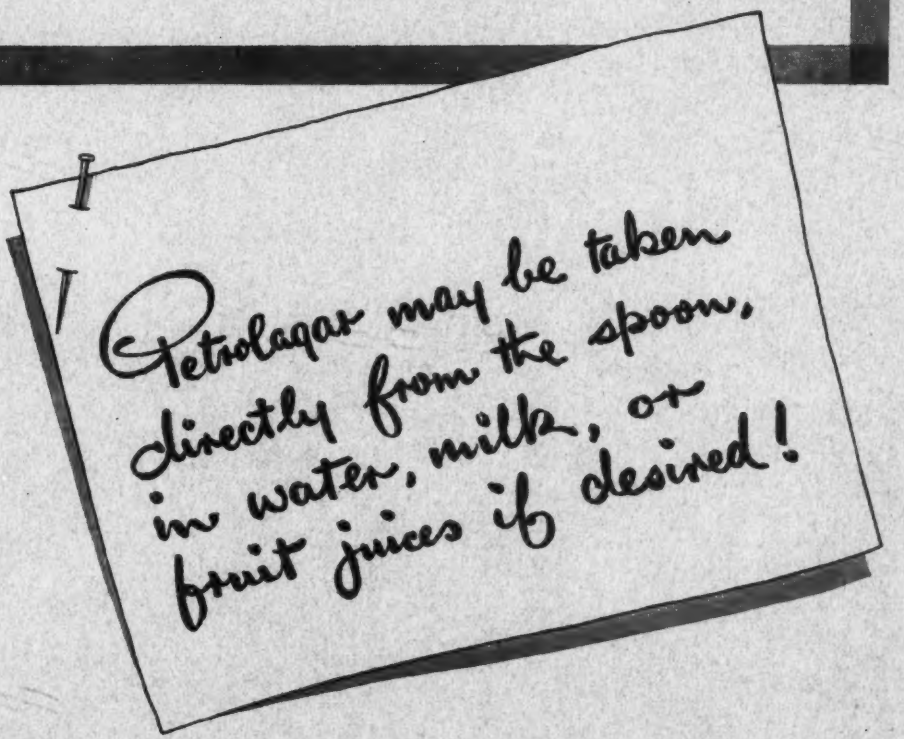

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HEART DISEASE (Symposium)

SOME OBSERVATIONS ON CORONARY OCCLUSION

R. H. Knowlton, M. D.
St. Petersburg

Dr. James B. Herrick in 1912 and again in 1918 brought the matter of coronary occlusion to the attention of the medical profession. Since then every aspect of the subject has been covered by so many excellent papers, some of them by members of this Association, that one may well question the advisability of any more. If one thinks that he has found something new on the subject, it is well to consult the earlier literature before announcing the fact. Still, there are points which can well be emphasized for, in spite of all that has been said and written, the diagnosis is at times difficult and errors of commission and omission have been made by the most enlightened.

Two preliminary statements might be made regarding the nomenclature; first, that the symptoms of coronary occlusion are those produced by myocardial ischemia (usually followed by necrosis) due to the blocking of the artery supplying the part; second, that myocardial infarction may also be caused by coronary narrowing without actual closure of the vessel. This is especially true when the circulation is further embarrassed by other conditions, such as pulmonary embolus, aortic stenosis or severe anemia[1]. With this understanding, the original term of coronary occlusion is used in this paper.

Pain in the chest occurring in women has always given me less concern than similar pain in men. I have also been impressed by the fact that coronary occlusion is relatively infrequent in women but that when it does occur the prognosis is distinctly worse than in men. Levy and Boas[2] found that coronary artery disease is about five times more frequent in men than in women. Willius[3, 4] in commenting on this discrepancy, suggested an interesting explanation based on the observations of Leary[5] in 1934 regarding the role played by disturbed lipoid metabolism in producing coronary artery disease. Supplementing this basis by results of his own studies, he suggested that there is some reason to believe that women are endowed with a more nearly perfect lipoid metabolism than men and can dispose of excess lipoid without depositing it in the arterial walls.

In cases of the acute coronary occlusion with its terrific pain, pallor, sweating and shock the condition is readily recognized. There are, however, other instances in which the closure appears to come on gradually, the symptoms are much milder and in which, in fact, there may be no pain at all. In such cases the physician is called to see a patient with prolonged substernal pain not following effort and often occurring at night. The symptoms suggest that a coronary occlusion has taken place, but subsequent events show that such is not true. These attacks of pain may be repeated over a considerable period, and when at length a complete closure does occur the patient may note little difference from the pain in previous attacks. At this time, however, slight fever and leukocytosis as well as definite electrocardiographic changes are usually noted. In this sort of case the physician has to use his best powers of persuasion in order to keep the patient in bed, because he may feel perfectly well and be free from the tormenting pain. It would appear that the muscle is now dead and gives rise to no more pain impulses, just as when the nerve in an aching tooth dies, the pain ceases.

Much has been written about the painless occlusion; undoubtedly it does occur and is many times unrecognized. One case was observed in which an unexplained rise in temperature in an anginal subject was the first clue to the diagnosis. One must realize, however, that people differ considerably in regard to their concept of pain, as well as in their sensitivity to it. Some may have a feeling of weight or oppression in the chest which they do not call pain. There may be pain equivalents, such as a choking sensation or a feeling of constriction in the neck, sudden dyspnea or inexplicable congestive failure[6].

Acute coronary occlusion with myocardial infarction may be entirely painless for quite

A symposium on Heart Disease was presented before the Sixty-seventh Annual Meeting of the Florida Medical Association, held at Tampa, April 29, 30, and May 1, 1940. It comprised the first three articles published in this Journal.

another reason, namely: that the patient is under full surgical anesthesia at the time of the attack. The following instance was observed last year:

The patient was a 60 year old woman without known heart disease. She was undergoing a spinal fusion, a procedure which would not be expected to produce any great amount of shock. Toward the latter part of the operation, it was noted that the patient was doing badly. The anesthetist stated she had been all right up to a moment or two before, when suddenly the respiration became poor, some cyanosis appeared and the pulse became weak, rapid and irregular. The condition was assumed to be surgical shock; oxygen and the usual stimulants were given. The operation was completed; the patient was put to bed in fair condition, and the treatment for shock was continued. During the next two days her condition did not change appreciably; the pulse continued to be weak and rapid, and the blood pressure remained at a low level, (90 systolic, 60 diastolic). On this account and because of the fact that the patient did not appear to be in surgical shock, a coronary occlusion was eventually suspected. On the third operative day an electrocardiogram showed a low voltage and a tracing typical of a posterior infarction. Ten days later the patient showed symptoms of myocardial failure, from which she recovered satisfactorily with the aid of digitalis; she was eventually discharged well as far as the cardiac condition was concerned.

One may speculate as to whether similar coronary accidents may not occasionally occur on the operating table and whether some cases of sudden death or of so-called surgical shock may not in reality be cases of coronary occlusion. Master[7] reported 35 instances of postoperative coronary occlusion which were not associated with any particular type of anesthesia. He considered of special significance the fact that in the medical wards during the same eight years only 4 cases of coronary occlusion occurred in contrast to the 35 and probably more in which the condition followed surgical procedure. Brumm and Willius[8], on the other hand, in observations on a series of 257 patients with severe coronary disease, who underwent necessary surgical procedures, noted that only 11, or 4.3 per cent, died from cardiac causes.

Coronary occlusion simulates a great variety of conditions in other organs, making the diagnosis at times a difficult one. Often the persistent nausea and vomiting commonly observed suggest an acute gastric disorder, which in itself may make a patient violently ill and cause a drop in blood pressure. The differential diagnosis sometimes cannot be made at once, especially in the cases of those patients known to have heart disease. Only subsequent events may distinguish between the two. Coronary occlusion used to be known as acute indigestion, but one must not make the mistake of calling all gastric disorders coronary thrombosis.

When the pain radiates downward, coronary occlusion may simulate various abdominal conditions such as perforated ulcer, pancreatitis or disease of the gallbladder. Resemblance to the latter condition is further increased by the occasional occurrence of jaundice, and in one instance I have noted bilirubinuria. In that case the early appearance of a pericardial friction rub made the diagnosis clear. Acute temporary enlargement of the liver may be confusing if one does not remember that it is an occasional finding in association with coronary occlusion.[9]

Sometimes the symptoms are referred more particularly to the lungs,such anomalies occurring as acute dyspnea, especially at night, rales in the bases of the lungs or even acute edema. Dr. Pincoffs[10] has called attention to the fact that coronary occlusion should be suspected in cases of atypical bronchitis and pneumonia, when disproportionate changes in the blood pressure or unusual dyspnea occur. It is stated that in a large educational hospital the condition most commonly diagnosed inaccurately was pulmonary infarction. The signs and symptoms of this abnormality may sometimes so closely resemble those of coronary occlusion as to make the diagnosis puzzling. However, a mistake does no great harm since the treatment of the two conditions is nearly identical.

Now that physicians have become "coronary conscious", it is probable that the diagnosis of occlusion is made far too often. Dr. Herrick[12] recorded some 30 conditions that he had seen mistaken for acute coronary thrombosis, and among them were the various forms of arrythmia. That a coronary accident may initiate an attack of acute auricullar fibrillation is well known, and it has been said that one may safely treat such a paroxysm in a patient with arteriosclerotic heart disease as though the attack were due to coronary thrombosis, until it is proved otherwise[13]. However, the fibrillation itself in an anginal subject may closely imitate an acute coronary closure, as illustrated in the following case report:

A man known to have coronary sclerosis and moderate intraventricular block was brought into the hospital suffering intensely from prolonged substernal pain. He was pale and sweating and had a rapid and irregular pulse, together with a lowered blood pressure. The condition was judged to be acute occlusion; treatment with morphine was given and repeated and oxygen administered without appreciable benefit. Aminophylline and dextrose were then given intravenously with almost dramatic relief of the pain. At the same time the fibrillation stopped, and the pulse became regular and remained so.

There was no subsequent fever, leukocytosis or change in the electrocardiogram to indicate coronary occlusion.

It cannot be said definitely that the aminophylline stopped the fibrillation, but it does seem evident that the fibrillation with the consequent inefficient filling of the coronary arteries was responsible for the pain, which disappeared when normal rhythm was resumed. Two other such cases have been observed in both of which the condition was coronary artery disease: in one the patient was an elderly woman who had a severe pain at every onset of fibrillation. White and Camp[14] in 1932 reported 3 cases in which definite prolonged attacks of anginal pain requiring morphine for relief were brought on by paroxysmal auricular fibrillation.

Many people who present themselves for treatment complain of having recently suffered from a precordial pain; in their cases one would like to rule out the possibility of a coronary accident. It is known that fever and increase of leukocytes are transient symptoms and that either one may be absent[15]. It is not practicable to take an electrocardiogram in every case and, besides, a single negative tracing does not rule out coronary thrombosis. The sedimentation rate, on the other hand, is practically always increased after the first few days and remains rapid for some time. The determination of that rate is a useful test, for a normal rate is good evidence that there has been no recent occlusion, and it can be easily made in the office without elaborate apparatus[16, 17]. It has also been frequently observed that the sedimentation rate offers a reliable index of the healing process of the infarction and that in each instance the patient should be kept in bed until his condition becomes approximately normal[18], or until there is no further change. It is well to make the test as early in the disease as possible in order that the patient's normal rate may be established before necrosis sets in. Otherwise the test is of less value, since patients with various pathologic conditions, especially rheumatoid arthritis, have a constantly increased rate.

The treatment of acute coronary occlusion is fairly well standardized: absolute rest, oxygen and morphine. Practically all authorities advise the free use of morphine to combat the terrific pain, and no doubt at times large amounts may be necessary to give relief. While the drug is indispensable in most cases, there are times when the symptoms are so mild that only one small dose is needed. Years ago before the knowledge about and recognition of coronary occlusion had penetrated to the rank and file of physicians, there were those who preached that morphia was a dangerous drug in the treatment of angina pectoris and apt to cause sudden death. It is now known that the physician rarely observes a case of true angina pectoris because the pain has gone before he arrives. The prolonged pain formerly called angina pectoris was in reality coronary occlusion, from which the patient frequently dies whether he is given morphine or not; cases have been observed in which the patient, although at last completely relieved of his suffering by morphine, unexpectedly died. It is not necessary to abolish the last lingering sense of discomfort. Wolferth[19] stated that the use of morphine is sometimes overdone; he suspected, he said, that the last unnecessary dose precipitates death. G. K. Fenn[20] also suggested that for aged patients the dosage of morphine be kept at a minimum by the use of papaverine and atropine. After the pain is relieved, the restlessness can be dealt with quite as well by phenobarbital sodium as by morphine.

Finally, the physician should have an optimistic outlook regarding coronary occlusion and should assure the patient that it may be quite possible for him to continue a successful and useful life. It is a serious mistake to instill in him the fear that another attack will mean sudden death, particularly when the diagnosis may be mistaken. Hearts may be damaged beyond repair, in which event death occurs shortly, but the younger and more vigorous hearts continue to go on functioning through the years.

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CORRELATING HISTORY, CLINICAL AND ELECTROCARDIOGRAPHIC FINDINGS IN THE DIAGNOSIS OF CORONARY OCCLUSION

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The use of instruments of precision to determine the site at which pathologic changes are taking place and the extent of the disease is to be encouraged, particularly when the condition may be as obscure as coronary occlusion. It must never be overlooked, however, that the use of delicate instruments requires considerable care and the interpretation of findings necessitates special training on the part of the operator if any degree of accuracy is to be attained. I think that at times some of these newer methods of diagnosis are not unlike Pandora's box.

The electrocardiogram has been of inestimable value in teaching us about disease of the heart, at first with relation to nerve conduction of the heart muscle and in more recent years with reference to occlusion of the arteries of the heart.

In some cases, as I shall illustrate, the history and physical examination and a single electrocardiogram will be sufficient to make a positive diagnosis. In other instances the electrocardiogram is only confusing. It must be remembered in dealing with coronary occlusion as with all other diseases that the history and clinical findings are of first importance; that if electrocardiographic tracings are solely relied upon, they may cause many difficulties. I shall attempt to illustrate my various points with clinical history, physical findings, and electrocardiographic tracings.

Read before the Sixty-seventh Annual Meeting of the Florida Medical Association, held at Tampa, April 29, 30, and May 1, 1940. (See footnote, page 177).

CASE REPORTS

Case 1. L. E. was a 48 year old professional man. His father died of cerebral hemorrhage at the age of 76; his mother died of cardiac dilatation, at 63. His personal history did not contribute information of importance.

In the late afternoon of January 6, 1938, the patient left his office and was getting into his car to drive home when he experienced a sudden pain across the lower anterior portion of the chest, slightly more pronounced on the left than on the right. There was an intense desire to belch. As he was passing the hospital the pain became so severe that he felt he could not go on and came into the waiting room. I saw him and placed him in bed immediately. The pain was intense and was only partially relieved by morphine. Altogether it continued for several hours. When the patient was admitted to the hospital the blood pressure was 210 systolic, 130 diastolic, and it continued at these points for six hours, when the systolic pressure began varying from 190 to 200. The following morning the blood pressure was 155 systolic, 95 diastolic. The patient had marked abdominal distention which lasted for five days.

The first electrocardiogram was taken on the morning of January 7, the day after admission. The interpretation of this tracing was that in spite of the slight inversion of lead 2, the tracing was not characteristic of coronary thrombosis even though such a diagnosis had been made clinically. A second tracing was made on January 10, which was interpreted as follows: "Marked change over the previous tracing, making coronary occlusion almost certain, though there are many things which keep this from being a typical coronary thrombosis." Noticeable changes had taken place in the T wave in lead 1, also in the T wave in lead 4 which was now markedly inverted. The next tracing was made February 3 when the QRS 1 was slightly notched, T 1 and T 2 less deeply inverted, T 3 less wavy, and T 4 of lower amplitude. The tracing made on March 14 indicated a return toward the normal. The last tracing made on May 6 showed still further change toward the normal. The T wave was still slightly inverted in lead 3.

Comment. This patient had sudden pains across the chest which lasted approximately six hours and were only partially relieved with opiates. There was marked distention of the abdomen with passage of blood from the kidneys. (The sedimentation rate markedly increased; the white blood count which was normal on January 7 and 10, was 9,500 on January 11) Serial electrocardiograms were necessary for diagnosis.

Case 2. A. C. K., a 62 year old physician came in for examination because of hypertension on May 3, 1938. His father had died of paralysis at the age of 57; his mother had died of paralysis when 68 years old. On examination the blood pressure was 192 systolic, 104 diastolic. At the request of this patient an electrocardiogram had been made the previous March 31 which showed an essentially normal tracing. Being a physician he did not get back for the physical examination he had requested until May 3.

The patient was admitted to the hospital at 10:20 a. m. March 11, 1939. He had been experiencing some substernal pain for the past two or three days and this had become severe in the early morning of the day of admission. He was more comfortable when he entered the hospital. An electrocardiogram was made a short time after admission which was interpreted as follows: "There are marked changes since the previous tracing. RT 1 is depressed and T 1 is diphasic. RT 2 and 3 are markedly elevated. RT 4 is markedly depressed. These changes are quite dramatic and probably represent a severe acute coronary thrombosis." The patient died at 2:20 o'clock in the afternoon.

Comment. This was a case of hypertensive heart disease over a period of several years. The tracing taken just one year before death was essentially normal.

Case 3. H. M., aged 47, was admitted to the hospital at 9 p. m., March 31, 1939, when his blood pressure was 150 systolic, 110 diastolic. He had had diabetes for several years; he also had hypertensive heart disease and angina pectoris. Prior to admission to the hospital he had suffered from acute pain similar to the attacks of angina but not relieved by nitroglycerin or by the usual means of treatment. Morphine was administered from which he received relief. The blood pressure the following morning was 100 systolic, 80 diastolic. A tracing indicated definitely an acute coronary thrombosis of the left coronary artery, anteriorly.

Comment. With this history, a patient of this age, and a tracing so definite, a diagnosis could be made from a single electrocardiogram.

Case 4. Dr. G. V. M., aged 86, entered the hospital on September 23, 1939. The first tracing in our series which was made in April 1927, when the patient was 74, was within normal limits. A second tracing, made in June 1929, when he was 76, was also normal. The night before admission, on September 22, 1939, the patient had suffered from severe pain in the chest. A tracing made two days after he entered the hospital was interpreted as follows: "1. Auriculoventricular heart block; 2. coronary heart disease." A further note was made that this tracing did not indicate a coronary thrombosis and that another tracing should be done in a few days. The next tracing, made three days later, indicated definitely an acute coronary thrombosis. Decided changes were noted in leads 1, 2, and 4.

Comment. The patient's age and period elapsing between the onset of symptoms and the time the tracing became positive were taken into consideration in making this diagnosis. The blood pressure was 85 systolic, 55 diastolic on admission, and 128 systolic, 78 diastolic, on April 28 when the patient was discharged.

Case 5. W. P. H., a 40 year old American ship's mate, was first seen in Riverside Hospital on July 4, 1939, at which time he complained of precordial pain of several hours' duration, followed by collapse, weakness, and profuse perspiration. The blood pressure was 155 systolic, 90 diastolic; temperature 97.2 F., pulse rate 82; respiratory rate 15. The patient was hospitalized and given general supportive treatment with morphine as needed for relief of pain. On the second day in the hospital the temperature rose to 100.2 F.; there was some congestion at the bases of both lungs; the blood pressure was 150 systolic, 90 diastolic. He had a low grade fever for several days and on the seventh hospital day the blood pressure had fallen to 112 systolic, 75 diastolic. He was kept at complete rest for four weeks after which time he was allowed to sit in a chair for a short time each day. The clinical diagnosis was coronary occlusion.

A tracing made on July 5 showed changes from the normal which were probably acute but not typical of coronary thrombosis. There was sino-auricular tachycardia. The next tracing, made on July 10, proved helpful. On this tracing RT 1 was less depressed, RT 3 less elevated, and RT 4 less depressed. The pulse rate was now 94 as against 116 at the time of the first tracing. These changes which had occurred since the first tracing were minor but definite. They suggested an acute cardiac process.

On the third tracing, made July 25, there were marked changes as evidenced by the deep inversion of T 2 and T 3, both being Pardee in type. These changes were now characteristic of coronary thrombosis with occlusion of the right coronary artery. This being a posterior infarction the prognosis was somewhat better than it would have been in anterior infarction.

A tracing made on August 9 showed that some changes toward the normal had taken place since the previous tracing. T 2 was not so deeply inverted.

Case 6. Mrs. W. F. S., aged 64, was first seen on January 6, 1938, at which time she complained of pain in the epigastrium and loss of weight. The blood pressure at this time was 134 systolic, 80 diastolic. The physical examination did not yield observations of importance. At this time an electrocardiogram showed a left axis deviation; otherwise the tracing was within normal limits. The patient was seen again on February 15, 1940, when the blood pressure was 175 systolic, 104 diastolic. She was having precordial pain from time to time on exertion and she was not sleeping well. A tracing was made on March 18, 1940 which was interpreted as follows: "Since the previous tracings there have been slight changes. Tracing is indicative of coronary disease but there is no evidence of coronary thrombosis." The next tracing was made March 23, 1940 when it was noted that there were no essential changes from the one previously made. The patient was admitted to the hospital on April 12 after having had four attacks of severe anginal pain during the night. Pain was relieved by nitroglycerin. She was apprehensive and exhausted. The blood pressure was 180 systolic, 95 diastolic, but during an attack it rose to 195 systolic, 105 diastolic. On the morning of April 16 she suffered a severe attack of anginal pain which lasted several hours and was only partially relieved by opiates. There was considerable shock. The temperature was normal on the morning of the 16th, pulse rate 98, respiration irregular but charted as 14. The blood pressure had dropped to 100 systolic, 70 diastolic. Because of shock no effort was made to take a tracing on this date. On April 17 a tracing was made with the following interpretation: "Since the last tracing two days ago there have been marked changes, chiefly in the QRS complexes, RT segments and T waves. QRS 1 is of much lower amplitude and is slurred. QRS 2 is iso-electric. RT 1 is elevated. RT 2 is slightly elevated. RT 3 is very slightly depressed. RT 4 is markedly elevated. T 1 and T 2 have a very slight late inversion. These changes are indicative of an early thrombosis probably of the left coronary artery (anterior infarction). Repeated tracings are desirable."

The patient's condition gradually grew worse. On April 17 the temperature rose to 102.2 F. and the blood pressure dropped to 80 systolic, 60 diastolic. A tracing made on this date was interpreted as follows: "There have been further changes since the tracing of 4-18-40. QRS 1 is widened and more slurred. QRS 2 is widened and notched. QRS 3 is more deeply inverted and widened. RT 1 is slightly more elevated and T 1 is becoming more inverted and Pardee in type. RT 2 is more elevated and T 2 is more upright. RT 4 is still greatly elevated and T 4 is somewhat more rounded and slurred. This tracing further confirms the diagnosis of acute coronary thrombosis of the left coronary artery (anterior infarction)." The temperature continued to rise and the patient died late that same day.

Comment. This patient was seen approximately two years before her death when the blood pressure was normal and there were no other findings of importance. The first symptoms of any serious coronary changes did not occur until in February 1940. Apparently the electrocardiogram picked up the changes as soon as they were evident clinically. When the patient entered the hospital, there was a definite question in our minds as to whether she had had a coronary occlusion before admission. The clinical examination did not suggest this and neither did the tracing which was made on the day of entrance.

Case 7. J. R. McG., a 53 year old ship's captain came to the hospital at 3 p. m. January 1, 1939. At 6 p. m. the day before he was seized with pain in the abdomen just

below the xiphoid process, extending to both shoulder blades. There seemed to be a viselike pain up against the heart, but no true precordial pain or pain in the left arm. The patient took about a quart of hot soda water without relief. Feeling bloated, he tried to belch but this did not lessen his distress. Hot salt water caused him to vomit and he obtained some relief. He had pain throughout the night and could not take a deep breath. On his admission to the hospital the temperature was 98 F., pulse rate 80 respiratory rate 18, blood pressure 120 systolic, 80 diastolic. The physical examination did not yield any other observations of importance. The blood studies were entirely negative; the icterus index was normal, and the stool negative. The sedimentation rate was 8 mm. in thirty minutes, 18 mm. in one hour. The following comment was made by Dr. Merritt after the history had been taken and the physical examinations made: "The history here is fairly clear-cut, though one should be on his guard for coronary thrombosis." I saw this patient with Dr. Merritt and Dr. Drew, who referred him, on the morning after admission. We concurred in Dr. Merritt's original opinion but concluded that coronary thrombosis must be ruled out. An electrocardiographic tracing was made on the morning of January 3 with the following interpretation: "The slight changes in P 2 and T 2 and the slight prolongation of the PR and QT interval make this a borderline tracing. They are suggestive of early arteriosclerotic changes. I do not believe that they represent acute changes, and this tracing is not indicative of coronary thrombosis." This patient was discharged from the hospital on January 3 after having been given a diet and careful instructions as to his future activity. He returned on January 18 for another tracing which indicated no changes from the previous one. This patient had early arteriosclerotic changes.

SUMMARY

Histories with brief clinical data and an explanation of electrocardiograms have been given for 7 patients. These indicate that:

1. Several days may elapse after there is clinical evidence of a coronary occlusion before the electrocardiogram gives any evidence of its occurrence.
2. The electrocardiogram may be positive within a few hours after the accident becomes evident.
3. There is still an occasional case in which a condition simulating coronary thrombosis occurs which may be due to acute gastric disorder caused by cholecystitis or cholelithiasis.
4. The electrocardiogram may, as in one of these cases, be a good indication as to the prognosis; on the other hand, it may be of little value.
5. The electrocardiogram may show changes as early as the clinical symptoms; again the symptoms may precede the electrocardiographic evidence by several days.
6. Finally, the history and clinical findings should be considered along with the electrocardiogram and a diagnosis should not be based on an electrocardiogram alone.

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THE AFTERMATH OF CORONARY OCCLUSION

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The clinical features of acute coronary occlusion have been so often reiterated in the medical literature in recent years that most physicians are able to diagnose the condition in the average case, but the *subsequent* clinical status of such patients is not so clearly defined in some quarters, which warrants the present brief review.

PATHOLOGY

Occasionally acute coronary occlusion is not accompanied by infarction in the myocardium, and in such instances there is no residual cardiopathy other than the obstruction in the involved artery. Ordinarily, however, acute occlusion is attended by myocardial infarction and the extent of the pathologic changes found in the heart, months or even years after the healing has taken place, depends on the magnitude of the area of infarction. A small infarct in the presence of ample collateral coronary circulation may leave no visible scar, only microscopic areas of fibrosis being found in the myocardium. A large infarct will leave a scar of considerable size which may in some instances become so thin and stretched that a saccular dilatation may be formed and, especially during systole, there may be definite bulging of such a scar beyond the line of the epicardium. This is called a cardiac aneurysm, and its site is usually at the apex of the left ventricle. Pericardial adhesions may cover the aneurysm lending support to the myocardium at this point, and sometimes thrombi form within the aneurysmal sac. Rupture of a ventricular aneurysm occurs rarely, much less often than rupture of the myocardium during the acute stage of infarction. If the infarct involves the interventricular septum, the auriculoventricular bundle may be permanently injured.

SYMPTOMS AND SIGNS

Surprisingly often, patients become symptom-free following recovery from acute coronary occlusion, and can take an active part in life for a varied period of years until they encounter another attack. As time goes on, however, most patients are constantly or intermit-

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tently reminded of the injury which the heart has suffered in several ways. Substernal or precordial pain is frequently noted. This may be so mild that it is frequently ignored, except in those whose attention has become riveted on the heart because of a former attack. Yet such vague pain may have real significance and warrants due clinical respect. At times there may be only precordial aching, or aching in the arms (chiefly the left) or in the interscapular area; or the substernal pain may be referred to these sites or even to the upper part of the abdomen. Whether mild or severe, the discomfort is usually induced by physical exertion, particularly walking; on the other hand, it may come while the patient is at rest and may even awaken him from sleep. Pain is sometimes provoked by a hearty meal and it is more easily induced by exercise if the patient has recently eaten. Emotional strain or discord of any sort tends to bring distress, and in some persons is the prime exciting factor. The pain is usually of brief duration, but in some instances it tends to drag on all day, once it has been initiated, only partly dulled by nitroglycerin or other medication. Full blown terrifying attacks of angina pectoris may develop, attended by angor animi, severe sweating, and weakness, but in my experience such classic attacks are observed rather seldom. If severe angina precedes the acute occlusion, the attacks may be milder afterward, although not necessarily so.

Fatigability is a common complaint and occasionally is the sole symptom during the "coronary aftermath". It is noted that tasks formerly performed without fatigue are accomplished only with effort. The voice tires easily and the mere presence of companions sometimes seems exhausting, even when the conversation is not exciting.

Palpitation may be noted particularly after exertion and must not be confused with the heart consciousness of the cardioneurotic patient.

Abdominal discomfort is complained of frequently and is described as a feeling of fulness in the upper part of the abdomen or a lingering uneasiness in the epigastrium. Belching often brings relief of this distress as well as of the more definite substernal anginal pain, so it is natural for the patient to focus his attention on the abdomen rather than the heart. That the condition is related to the heart is suggested by the aggravation of the abdominal complaint by exercise after eating or by emotional factors. Pain simulating that of peptic ulcer occasionally develops which may prove confusing; since peptic ulcer sometimes co-exists, such cases need careful clinical study. It is worth remembering, however, that when pain of this character arises for the first time during the "coronary aftermath" it will likely prove to be of anginal character.

Insomnia is sometimes troublesome, particularly in those patients who have not made proper mental adjustments to the heart disease. Insomnia is seldom due to angina decubitus, but in some instances it may be due to the same mechanism as paroxysmal nocturnal dyspnea.

Personality changes develop in some patients following coronary occlusion, the family or friends noting petulance and irritability or moroseness. Or there may be only indecision, a loss of power of concentration or a vague unrest of spirit, with lack of interest in any aspect of life. Intolerance for cold may become quite marked.

Congestive heart failure may ensue at any time during the "coronary aftermath", differing in no way from failure due to other types of heart disease. If the failure is predominantly in the left ventricle dyspnea on exertion, orthopnea or paroxysmal nocturnal dyspnea and cough will prevail; the vital capacity will be lowered and tolerance for exercise reduced. Dependent edema and possibly hydrothorax and ascites appear when the right ventricle fails, although an increase in venous pressure may be the only clearcut sign of early right sided failure. Even though improvement may follow proper therapy, one should expect recurrence of congestive failure after an interval of weeks or months.

Physical findings of diagnostic significance are more often than not lacking in patients who have recovered from the acute phase of coronary occlusion. In the absence of complications the most common abnormalties noted are weak heart tones and moderate hypotension, but such findings are never very significant. Cardiac hypertrophy and dilatation may be found particularly if multiple infarctions have developed or if hypertension is present, and a systolic apical murmur due to dilatation of the mitral ring is audible frequently. If a cardiac aneurysm

has formed the diagnosis may be difficult, although Libman[1] stated that a pulsation distinct from the apical pulsation and accompanied by a dull first sound and gallop rhythm signifies aneurysm. The radiologic diagnosis of cardiac aneurysm is often difficult. According to Berk[2], if the aneurysm is diffuse the apex is dilated eccentrically with systolic contraction hardly visible; if it is circumscribed a definite bulge is seen, and there may be paradoxical pulsations. Kymography is an aid in securing an accurate diagnosis of cardiac aneurysm, as well as in depicting areas of healed infarction in the ventricles not shown by ordinary radiologic examination. For details of this examination, the papers by Hirsch[3] and, more recently, by Sussman, Dack, and Master[4] may be consulted.

Electrocardiography may disclose no abnormalities but commonly there is some relic of the acute changes which occur in myocardial infarction; or frequent premature beats, auricular fibrillation or varying grades of auriculoventricular block may be found. In other instances bundle-branch block shows up; in fact one has a right to suspect a previous coronary occlusion whenever evidence of this abnormality appears in the electrocardiogram.

COMPLICATIONS

A condition simulating subdeltoid bursitis or periarthritis of the shoulder (usually the left) is a common complication which follows coronary occlusion. The shoulder is painful, almost continuously so, and is made worse when abduction or external rotation is attempted, or when the patient lies on the affected side. Painful swelling of one or both hands may add further distress. Although this complicating pain of the shoulder has been described by a number of authors, including Edeiken and Wolferth[5], Libman[6], and Boas and Levy[7], some clinicians still confuse it with referred anginal pain, and keep those afflicted in unnecessary confinement for long periods of time. Numerous theories have been presented as to the underlying cause, but the exact mechanism producing the painful shoulder syndrome remains obscure, both metabolic and neurogenic factors probably playing a part. The condition usually persists for several months, sometimes for a year or more. In some instances there is a deposition of calcium salts in one of the tendons (usually the supraspinatous) about the subdeltoid bursa, or the calcareous material may rupture into the bursa with relief of pain, as recently pointed out by Weiland and Burbacher[8].

Delayed embolic phenomena may serve as complications even months after the acute occlusion, although most emboli from the mural thrombus underlying an infarcted myocardium are thrown off during the early days of the acute attack.

Peripheral vascular disease, chiefly arteriosclerosis involving the vessels of one or both legs with intermittent claudication, occurs fairly commonly in patients who have suffered coronary occlusion previously, and in some instances limitation of walking is due more to the peripheral vascular disorder than to the condition of the heart.

Disease of the biliary tract may be a distressing complication during the "coronary aftermath". If there is cholecysitis or cholelithiasis, attacks of anginal pain may be aggravated or even initiated, according to Fitz-Hugh and Wolferth[9] who demonstrated clinical relief of such pain following cholecystectomy. On the other hand, Levine[10] recently declared he had never seen true angina disappear after this surgical procedure. Peptic ulcer, as noted above, may co-exist, and if the pain from the ulcer follows a path similar to that of anginal pain, it may be impossible to differentiate between the two, except for the fact that alkali will relieve the one and nitroglycerin the other. If a malignant condition develops in the stomach or bowel, care must be taken not to attribute the resulting symptoms to the cardiac disease. If lesions develop in the urinary tract they can usually be diagnosed without confusing them with the cardiac disease.

TREATMENT

Many patients will not need any therapy during the "coronary aftermath" other than proper rest. Nitroglycerin is the most important drug used in alleviating anginal pain; it is given sublingually in doses of from 1/250 to 1/50 grain. As patients show a marked difference in tolerance to this drug, it should be used with due care. It may be used prophylactically before the patient undertakes any form of physical exertion, or before he attends the theatre or a social function. If the pain is relieved more satisfactorily by whiskey or brandy, there is usually no contraindication to alcohol in modera-

tion. At times morphine sulfate is required in the usual dosage. Self medication must be relied upon as it might prove dangerous to wait for a physician's visit. To reduce the frequency of painful attacks, one of the xanthine derivatives such as aminophylline, theobromine-sodium salicylate, or theocalcin should be tried. Although there is considerable argument as to the value of these preparations, there seems little doubt that some patients note fewer attacks of pain while taking one or another.

In recent years I have induced a number of such patients to install in their homes an apparatus* for the inhalation of oxygen as it has been shown that the use of 100 per cent oxygen for a few minutes will end most attacks of anginal pain. By having the oxygen immediately available, many patients have been rendered more comfortable, and some prefer it to the use of nitroglycerin. On some occasions it has proved extremely fortunate to have oxygen so readily at hand to combat attacks of acute failure of the left ventricle or a recurrence of coronary occlusion.

The usual therapeutic regime should be instituted if congestive heart failure supervenes: digitalis, diuretics, sedatives, and the restriction of fluids and salt.

Cheerfulness and encouragement are of great value to the patient who, with a natural lack of enthusiasm, is anticipating a recurrent attack of coronary occlusion.

The diet should be plain, the patient's weight should be kept within bounds, and constipation should be avoided by the usual measures. Sometimes a striking improvement in general health follows the use of vitamin therapy. If the appetite is poor and the patient is underweight and listless, the use of insulin as a tonic measure is justified. Care must be taken to supply abundant amounts of carbohydrates in the diet, thus avoiding hypoglycemia and its attendant risks. I have used insulin in a number of such cases during the past ten years, and it has not precipitated angina or coronary occlusion in any instance. I have found it to be a very useful measure in restoring some of these patients to better health[11]. It should not be used, however, by any patient who is not willing to follow orders carefully to avoid hypoglycemic reaction.

* The apparatus used for the administration of 100 per cent oxygen, is that designed by Boothby, Lovelace and Bulbulian, and the mask is termed the B.L.B. mask.

SUMMARY

A review of the common features of the clinical status which follows recovery from the acute phase of coronary artery occlusion, has been presented. For the sake of clearness and brevity many details making up the clinical picture have been omitted. Some comments on therapy have been added.

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DISCUSSION

Dr. W. C. Blake, Tampa:

During the last decade the laity as well as the members of the profession have become conscious of the increasing prevalence and usual symptomatology of coronary disease. With the trend in population favoring a relative increase in numbers in the later decades of life while at the same time the increasing complexity of human existence exacts its toll, the evaluation of the sufficiency of the coronary circulation has become one of the major problems confronting the internist. Coronary occlusion with myocardial infarction is one of the most common complications of coronary disease. Frequently its onset is dramatic, and a clinical picture is produced that could hardly be mistaken for any other condition. As Dr. Knowlton has pointed out, this symptomatology may, however, vary within wide limits, or the onset of gradual myocardial failure may furnish the only clue to its occurrence.

On the other hand, it is safe to say that the diagnosis of occlusion is made far too often on insufficient grounds. This error would not be made so frequently, as Haman and others have reminded us, if the other circulatory and extracirculatory conditions capable of producing anterior chest pain were kept in mind. In no other disease is a careful clinical history of such paramount importance

Dr. Cason has admirably set forth the importance of careful scrutiny and consideration of the entire laboratory and clinical pictures, if errors of commission or omission

are to be avoided. No single bit of evidence is in itself pathognomonic. Even the T wave changes so constantly associated with occlusion occur also at times with hypertension and both acute and chronic pericarditis. Again, the evidence often rests heavily on minor cardiographic changes. It should be borne in mind that a normal cardiogram may be obtained in the presence of known serious coronary insufficiency. Even after infarction has actually taken place, the typical cardiographic changes may not appear for a period of six or eight days. Consequently, if repeated examinations are not made where the clinical picture warrants it, the true condition may pass unrecognized. Such a situation is tragic, but it is also just as tragic to see a person condemned to a life of invalidism when no serious coronary insufficiency exists. Permit me to emphasize again the necessity of careful scrutiny of both the clinical and laboratory picture before a definite diagnosis is made, lest the etiologic diagnosis be missed.

Dr. Nichol has ably presented the problems pertaining to the aftermath of coronary disease. Not the least of these is cardiac neurosis. Extravagant publicity has made it well known to the laity that among the chief causes of mortality heart disease now leads with a tremendous majority, causing many more than twice the number of deaths attributed to cancer, its nearest competitor. One of the great advances of our generation has been the clear designation of the more important causes that may act separately or simultaneously to damage the heart, and of these causes coronary disease and hypertension are perhaps best known to the general public. In dealing with patients suffering from coronary disease, it is highly important to distinguish between immediate and ultimate prognosis. There is a tendency today to diagnose prematurely "permanent total disability" when the immediate prognosis is temporarily grave, for many times there is a strong possibility of recovery to a fair state of health with years of life ahead.

To win the intelligent cooperation of the patient and allay unwarranted fear of this cardiac ailment that strikes terror, often needlessly, is no small part of the physician's task. In this disease, common as it is among business and professional men of middle age today, some patients die in a few seconds in their first attack, but by far the most of them live a number of years, and a few survive twenty years or longer.

Dr. E. W. Bitzer, Tampa:

I have enjoyed the papers and think they deal with one of the most interesting subjects in the category of medicine. There are two things I would like to talk about to you for a few minutes.

One is the possibility of foretelling the onset of acute coronary occlusion. For many years an exercise test has been used in cardiographic work. In recent years Dr. David Scherf has become very enthusiastic about this test and has collected some interesting cardiograms showing the changes following exercise. Being interested in this test I tried it in a number of instances with very indifferent success and concluded that only in luetic occlusion of the coronaries was it of value. I think in those cases it is very interesting.

Some years ago I conceived the idea that perhaps producing certain changes in the blood pressure might lead to significant changes in the cardiogram. With this in view I subjected a number of patients suffering from angina pectoris to the cold pressor test, developed by Brown and Hines. In some patients there was a jump of 80 or 90 mm. in the blood pressure, but in less than 1 per cent of them was there any change in the cardiogram which was taken during the time when the blood pressure was elevated.

I had one very interesting experience while doing this. One woman who had a hypertensive cardiac condition was evidently supersensitive to cold. She had found that putting her hands in cold water or handling ice would immediately precipitate an attack of angina pectoris. She also found that putting her hands in warm water would relieve the attack. In this instance there was a prompt onset of pain in the chest when the hand was immersed in cold water. There was also a jump in the blood pressure of about 35 mm. Later on I gave carbon dioxide inhalations and produced the same jump in pressure but this did not precipitate an attack.

Some day I believe we will work out a means of determining coronary disease before the onset of symptoms and this will be of great advantage.

The other condition about which I want to speak is the various forms of arrhythmia in coronary occlusion. The most common form is premature beats. This, however, is relatively unimportant. Heart block has already been mentioned and, of course, bundle branch block is fairly common. The uncommon ones are the tachycardias, which create a very embarrassing situation when they occur in the course of acute coronary occlusion. Recently I observed one such case in which the pulse rate was 220. It proved to be ventricular tachycardia and death followed promptly. Auricular fibrillation may occur and is frequently fatal.

Dr. R. M. Harris, Miami:

There are one or two remarks I would like to make concerning prognosis. I think that the prognosis depends upon whose statistics we consider at the present time. In some series the mortality has run as high as 50 per cent, whereas in some of the more recent studies reported it has been as low as 10 per cent. In other words 90 per cent of patients under 40 years of age have a chance to survive their first attack of coronary thrombosis. The mortality of course goes up as the age of the patient increases. On the other hand, a young person who suffers an attack of coronary thrombosis has a definite alteration in his life expectancy.

An early correct diagnosis very definitely alters the prognosis in coronary thrombosis. Pain is not as important perhaps, as it was thought to be at one time. Of course pain lasting over a period of two or three days usually indicates a fairly widespread lesion and in that respect it is important.

Shock is extremely important. For in those patients who manifest evidence of severe shock the mortality rate is definitely higher, and for those who show circulatory failure and congestive heart failure the mortality rate may run as high as 70 or 80 per cent.

Dr. Knowlton brought out some statistics with regard to the incidence of coronary thrombosis in men and in women. I believe he said the ratio was approximately 5 to 1. I feel this is approximately correct although I believe that Dr. Levine in his recent series found the incidence to be perhaps as low as 3 to 1.

The existence of fever over a long period of time, a week or ten days, is of definitely bad prognostic significance. Further, a drop in blood pressure where the pulse pressure is 20 mm. or less is always a bad prognostic sign. This was brought out very clearly by Dr. Cason.

The existence of arrhythmias frequently causes concern. Sometimes it demands no treatment and sometimes it does demand treatment. It is the custom of one or two outstanding cardiologists to give routine doses of quinidine sulfate to patients with coronary occlusion who show definite signs of arrhythmia.

Dr. Thomas E. Daly, Palm Beach:

This subject has been so well covered that there is very little left for me to say, so I will pick up loose ends and add a little here and there to what has been said.

If I were to say nothing else I would have to stress the fact that the family and personal history in these cases is more important than any other one thing. Physical examination comes next in line and the electrocardiographic reading or diagnosis, if you wish to call it such, runs a poor third. In fact, it is just one more laboratory test as is the basal metabolism and the sedimentation rate. I think that one single electrocardiographic reading is hardly worth the paper it is developed on. Serial electrocardiograms, however, are of importance and of value.

Sometimes clinical symptoms will precede the electrocardiographic signs by as much as ten days. Sometimes signs will never appear on the electrocardiogram. It is well to bear these facts in mind.

The question of frequency of coronary occlusion or coronary thrombosis in women and men or comparison of the two arises: I have sometimes wondered whether the wearing apparel of men and women did not have some bearing on this, as was brought out at the recent meeting of the American College of Physicians. Women used to, and some of them still do, wear some type of abdominal supports and much lighter clothing than men. I believe that these are worthy of a try in treating these patients,—I am referring to the men now as well as the women. I have prescribed surgical belts for several patients in West Palm Beach and Palm Beach, and I think they are being benefitted to a great extent.

Not much has been said about therapy in the discussion today. That can be easily appreciated as there is not too much to be said. The most useful drug, if it can be called a drug, is oxygen. If the patient cannot be removed to a hospital, oxygen should be brought to the patient at home before anything else is done, and before everything else. Morphine is a helpful drug but it can sometimes be a harmful one if the patient has myxedema, for he may die suddenly.

Dextrose, I think, should be used only when there is dehydration caused by vomiting. Otherwise it may prove harmful.

Digitalis I mention just to caution against its use. Digitalis can be harmful and we have been too inclined, when patients had auricular fibrillation, to prescribe digitalis immediately. Digitalis won't help auricular fibrillation much in these cases, as it increases the work of the heart. If the patient is let alone auricular fibrillation will usually stop unless the lesion is so bad to begin with that it will cause death. However, when congestion sets in the doctor has no other alternative. He must use digitalis.

There has been much discussion about the use of quinidine for coronary thrombosis although it has seldom been reported. I often wonder if it is not of some value. Frequently I administer quinidine three times a day in small doses with the hope of warding off ventricular fibrillation as I believe many of these patients die from just that. The use of vasodilators is theoretically sound but not conclusively proved of worth.

Dr. Julian E. Gammon, Jacksonville:

From a pathologic point of view there are four distinct types of coronary disease. The first is perivascular fibrosis in which the vessels are to a certain extent insulated from the muscular tissue and this is the lesion in which we see cardiac dilatation and congestive failure. The cardiogram usually does not show evidence of disease excepting when arrhythmia is present.

The second type is commonly called hardening of the arteries and is due to deposits of calcium beneath the intima and in the muscularis which frequently narrows the coronary orifices and may gradually occlude the vessel at the opening into the aorta. In such instances collateral circulation through the thebesian veins and coronary artery anastomosis may give the heart a fair degree of efficiency. The arteriosclerosis due to calcium deposits is frequently associated with angina pectoris and the cardiogram may not show evidence of disease.

The third type is atheromatous degeneration of the intima and it is on this lesion that a clot may form and produce coronary thrombosis and occlusion of the vessel with infarction in the heart muscle. It is the infarction of the heart muscle which produces the characteristic cardiographic signs of sudden vascular occlusion from thrombosis. Should the occluded vessel cause infarction of the interventricular septum the condition is serious since the conduction pathway in the Tawara-His bundle or its right and left subdivisions are severely damaged or destroyed. The result is heart block or bundle branch block. There is also the possibility of intraventricular thrombi forming and secondary embolism. Embolism may also occur when the infarct involves the ventricular wall producing pericardial friction rub and thrombi on the endocardium. These patients have profound weakness and have marked drop in blood pressure. When the infarct is superficial, however, the blood pressure may not fall unless shock is produced. Some of the latter patients may pass as victims of angina pectoris and on coming to autopsy in a few years, the superficial scars of infarction may be seen. In such a patient the cardiogram may not show the characteristic evidence of the true condition. Hence, it is a wise plan not to depend too strongly on a cardiogram in evaluating heart disease for a diseased heart may show a normal electrocardiogram. Furthermore, one patient may have the characteristic cardiographic signs of infarction and die; another may be a chronic heart invalid and still another may return to his normal state of health and live a number of years. The layman may ask, "How else then can you tell a sound heart from a diseased heart?" A knowledge of etiology and pathology, together with clinical experience and the aid of instruments of precision may answer this question.

Time does not permit the discussion of the fourth type of coronary disease, endarteritis.

Dr. R. H. Knowlton, (concluding):

It was not my intention in this brief paper to cover more than a few of the varied features of coronary occlusion which might be of practical interest. These observations are in general supported by references to the recent literature with the exception of the occurrence of coronary thrombosis in an anesthetized patient. I feel sure that my associates join me in thanking the physicians who added to the presentation by their generous discussions.

Dr. T. Z. Cason, (concluding):

There are only one or two little points to go into.

First, despite the almost complete failure of my picture, I want to say that this is a very interesting and instructive way to learn something about the electrocardiograph.

Second. In spite of the fact that I have been studying electrocardiograms and watching them for a little over eighteen years I still do not know a great deal about them. I still find there is a tremendous lot to learn. None of us should get to the point where we think we know it all.

The younger men coming out today are learning something about the interpretation of electrocardiograms and something about the proper method of substantiating the findings in these cases. It is most interesting. If we older men do not start out right away these young fellows are certainly going to put it over on us.

Dr. E. Sterling Nichol, (concluding):

The question of cardiac neurosis was brought up. Out of a fairly large group, I have encountered about fifteen instances of combined cardiac neurosis and coronary thrombosis. It is not a pleasant experience, having assured a patient he has cardiac neurosis but a sound heart, to see him stricken down within a month or so. I have had that happen twice in the past few years. I observed one striking instance of coronary thrombosis, considered cardiac neurosis, a few years ago in a very healthy appearing, robust and energetic 37 year old man with a tremendous love of life and activity. Because of a previous addiction to morphine, when he developed pain, although considered at first coronary in origin, the cause was later believed to be cardioneurosis brought on by marital discord. This patient had severe seizures of pain without much evidence to show for it on clinical examination, so that one hardly could conceive his pain as being due to coronary insufficiency. Against my advice and that of other physicians in the North, he convinced one of the neurosurgeons in a large clinic that he should undergo sympathectomy for his cardiac pain. He actually chose that as a method of suicide, for he knew that he could not tolerate such an operation. He died of shock on the table. Autopsy revealed three large areas of fibrosis due to separate coronary thromboses in the past.

SURGICAL TREATMENT OF ESSENTIAL HYPERTENSION

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Miami

There is probably no type of case treated by the internist with a greater feeling of pessimism than that of the relatively young individual suffering from essential hypertension. The internist should, therefore, welcome the application of surgical treatment to this type of case.

It is generally conceded that the medical treatment of essential hypertension is only palliative. The usual medical treatment, consisting of rest, diets, and varied medications, may lower the blood pressure temporarily; but the final outcome is invariably the same. Adson[1] has classified hypertensive cases into three groups: slow, moderate, and rapid. He estimates the life expectancy in each group as 10 to 15 years for the slow; 3 to 4 years for the moderate; and 18 months for the rapid. At best, medical management of these cases results in total and permanent disability, so far as remunerative work is concerned; and unfortunately, the financial status of the majority of patients prevents them from availing themselves of the advantages which might be gained from rest and mental relaxation. In the cases of these individuals in the lower income brackets surgical treatment is especially intriguing, because it may enable the hopelessly disabled individual to resume gainful occupation within a short period of time.

In selecting cases of hypertension suitable for surgical treatment it is most important to have a thorough understanding of the pathological physiology underlying this disease. First of all, a very careful differentiation must be made between "essential hypertension" and hypertension of known organic etiology. In studying these cases we must first identify those which may be attributable to some known pathological condition such as hyperthyroidism, suprarenal tumor, pituitary disease, nephritis, and arteriosclerosis. In such cases the treatment should be directed towards the underlying disease. The etiology of essential hypertension has not been definitely established, but the outstanding work of Goldblatt[2] has at least afforded a tangible basis upon which to theorize. Goldblatt devised an ingenious metal clamp with which he was able to apply varying degrees of constriction to the renal arteries of dogs. He found that uremic symptoms and death occurred only when there was sudden, severe, bilateral shutting off of the arterial supply to the kidneys. A gradual, partial constriction of one or both renal arteries produced a renal ischemia and a permanent rise in blood pressure with a lowered urea clearance as the only indication of impaired renal function. It was further determined that the release of this arterial constriction resulted in the return of the blood pressure to normal. Subsequent experiments by Page,[3] Blalock[4] and many others have pretty well established the fact that when a kidney is deprived of an adequate blood supply it produces some "pressor substance" which raises the blood pressure above normal in an effort to regain an adequate blood supply for itself. This correlation between renal blood supply and essential hypertension has afforded a physiological approach to the surgical treatment of the disease. It is felt that many cases of essential hypertension may be relieved by correcting renal ischemia and reestablishing an adequate blood supply to the kidney.

Experience with surgery of the autonomic nervous system in connection with peripheral vascular disease has shown us that an interruption of the sympathetic nerve supply produces a vasodilatation and, consequently, an increase in blood supply. Following out this idea, one would be justified in assuming that the most practical way to increase the blood supply to the kidney would be to interrupt its sympathetic nerve supply; and this is the approach which has proved most satisfactory.

The surgical treatment of hypertension has been approached from several different angles. A good many years ago Crile[5] and DeCourcy[6] attempted to relieve hypertension by operating upon the adrenal glands. They believed that hypertension was produced by abnormal secretion of adrenalin. This idea has now been abandoned. Adson[7] performed extensive rhizotomies on individuals with hyper-

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tension, sectioning the anterior roots from the sixth thoracic to the second lumbar segments in order to produce a paralytic vasodilatation of the entire splanchnic area. This procedure was none too successful, and the associated mortality and morbidity were entirely too great to justify its clinical application. A little later Craig[8] developed a technique by which he sectioned the greater and lesser splanchnic nerves and at the same time removed the first lumbar ganglion and resected approximately four-fifths of the adrenal gland. His results in these cases were more encouraging but far from constant. The procedure was a rather formidable one, carrying with it a high mortality and requiring a two-stage procedure to do both sides.

In 1929 Peet,[9] using a supradiaphragmatic approach, first sectioned the greater and lesser splanchnic nerves bilaterally in an attempt to relieve gastric crisis. This experience convinced him that the supradiaphragmatic approach afforded a relatively simple and harmless method for the surgical interruption of the splanchnic enervation, and in 1933 he first performed this operation for the relief of hypertension. In 1935 he reported the results of his first 100 cases. Peet found that in this early series the results more than justified the slight danger encountered in doing this procedure. Frayberg and Peet[10] have shown that depriving the kidneys of their sympathetic nerve supply has had no undesirable effects, and in some cases there was an actual improvement in renal function after splanchnicectomy.

Peet and his associates have performed intrathoracic splanchnicectomies on well over four hundred cases. Many of them have shown a persistence of clinical cure or great amelioration of symptoms for as long as five years. The period of elapsed time since these operations have been performed is not long enough to permit any definite statements in regard to the permanency of good results. It is evident, however, that the results have been sufficiently encouraging to justify further use of surgery in the treating of selected cases of progressive essential hypertension.

Craig[11] and other surgeons at the Mayo Clinic have had similar experiences in their large series of cases and they are, for the most part, enthusiastic in the reports of their experiences in the surgical treatment of hypertension.

Crile[12, 13] is a most enthusiastic advocate of the surgical treatment of hypertension. In studying a large series of cases in which he had operated he found that eighty-seven per cent of the patients received symptomatic relief and thirty-five per cent were completely cured with a return of blood pressure to normal.

Heuer[14] and Page[15] have been more pessimistic in their reports, but even in their pessimism they state that they have observed patients with essential hypertension incapacitated and practically bedridden as a result of the disease, who have returned to their customary work for a period of as long as three years. They feel that this should be sufficient encouragement to justify the continuation of surgical treatment. Smithwick[16] is of the opinion that patients with essential hypertension that is shown to be definitely progressive in nature should be subjected to sympathectomy as early as possible, before advanced organic changes have taken place in the vascular system. He feels that the immediate outlook seems encouraging and time may show that the disease may be arrested, or even cured, if operation is performed before arteriosclerosis takes place.

As stated previously, the surgical treatment of hypertension has not been employed long enough to afford a fair evaluation of its possibilities; however, the experience of Peet[17] seems to have been shared by many others carrying out this type of treatment. Peet found that seven per cent of his patients showed no improvement; sixteen per cent showed slight improvement; twenty-nine per cent showed moderate improvement; thirty-three per cent showed marked improvement, and fifteen per cent seemed to be entirely cured. In other words, seventy-seven per cent of all his patients have received worthwhile benefit.

The selection of cases for this type of treatment presents a most difficult problem, and no definite critera have been established. The general opinion, however, seems to be that the patient should not be more than fifty years old; the systolic pressure should be 200 mm. or more; the nonprotein nitrogen

should not be above normal limits; there should be a urine concentration of 1.010 or more; the urea clearance should be above 30 per cent; the heart should be in compensation. There are certain clinical observations which aid us in selecting appropriate subjects for this operation. Hines[18] has shown that the effect of vasocontraction on blood pressure may be evaluated by placing the patient's hand in ice water. If doing this produces an appreciable rise in pressure, one may assume that vasospasticity is playing a part in producing the hypertension, and for that reason a fall in pressure may be expected following interruption of the sympathetic nerve supply to the splanchnic vascular bed. Any prospective candidate for surgery should be tested in this manner and should also be observed under varying conditions. If there is an appreciable fall in blood pressure when the patient is put to bed and given sedatives, one may assume that the same results may be attained permanently by surgery. If the pressure rises sharply under emotional stress or physical activity, one is justified in believing that such peaks in pressure can be prevented by interrupting the sympathetics.

The procedure which I use is the intrathoracic splanchnic section devised by Peet. This consists of resecting the paravertebral portion of the eleventh rib subperiosteally and retracting the pleura laterally. This affords an adequate exposure of the lower mediastinum and enables one to resect an ample segment of the splanchnic nerves and also remove the tenth, eleventh and twelfth thoracic sympathetic ganglia together with the intervening portion of the thoracic sympathetic trunk. The bilateral procedure may be done at one operation and requires from forty-five minutes to one and one-half hours. This can be done under local anesthesia, but I prefer a light general anesthesia. There is little or no danger to the patient, and the average patient is ready to leave the hospital in four or five days.

Following such a procedure the initial drop in blood pressure is spectacular. Frequently intravenous fluids have to be given to keep the pressure up to minimum requirements. Following this initial plunge the pressure slowly rises until at the end of two weeks or a month it has frequently regained its preoperative level. After the first month or six weeks there is a gradual reduction of hypertension, and after ninety days a new constant pressure is usually established at a gratifyingly lower level.

SUMMARY AND CONCLUSIONS

Experimental evidence indicates that essential hypertension is caused by renal ischemia. This renal ischemia may be corrected by depriving the kidneys of their sympathetic blood supply, and thereby producing a vasorelaxation of the renal arterioles.

Renal sympathectomy may be accomplished with a minimum amount of risk by sectioning the splanchnic nerves and the thoracic sympathetic trunk in the lower mediastinum. Individuals under fifty with no evidence of arteriosclerosis, nephritis, or other responsible organic disease who suffer from a progressive hypertension should be considered as prospects for surgical treatment. If their blood pressure undergoes a sharp rise when their hand is placed in ice water, and if there is a definite fall in pressure when the patient is put to bed and given sedatives, surgical treatment should be undertaken.

In advising surgery for such an individual he should be told that there is a 25 per cent chance of obtaining a complete cure; a 25 per cent chance of obtaining marked relief; a 25 per cent chance of obtaining some relief, especially from headaches and nervousness; and a 25 per cent chance that he will receive little or no benefit.

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ANESTHESIA

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In this paper I shall try to cover the topic of anesthesia in a general way. An understanding of the advantages and shortcomings of various anesthetics in common use will allow the clinician to judge the type of anesthesia to be used in any particular case. It will also enable him to evaluate the relative safety and scope of various methods, to form his own conclusions and appraise the claims of enthusiasts in various fields.

The prime requisite of an anesthetic should be dependability and consistency in its mode of action. It should be above all things safe. It should be easy to control and its actions should be readily and easily reversed. It should narcotize the patient rapidly without physical shock and should obtain complete relaxation for as long a period as is desired. Its withdrawal or the patient's awakening should be without unpleasant effects. It should produce no pathology, per se. It should not unduly affect the sympathetic nervous system, as neurological signs clearly defined and in recognized sequence are our chief guide to stages of anesthesia.

There is no anesthetic that in my opinion fulfils all these requirements. Some do one thing better than others. I believe that, all things being equal, granting an ideal patient, the most intelligent approach to the problem of selecting the best anesthetic would be first, since we cannot have everything, to decide which of the above factors are the most important.

I think you will all agree that safety is the foremost factor; then come dependability and ease of control, with ability to produce relaxation a close third. While the average patient wants and has a right to expect an anesthetic which is easy of induction, to my mind this factor should be the last to consider. The fact that an anesthetic produces no lasting pathologic changes in tissue organism, that it is safe and easy to control is more important than momentary discomfort which a patient may have either in going under or coming out of his anesthesia.

So let us examine various anesthetics which are commonly used in this locality to see how they fulfil these qualifications.

First, we will consider local anesthetics. These react by direct contact with the nerve endings. They are known by many names but are all synthetic derivatives of cocaine, such as novocain, scurocaine, procaine, gravocain, spinocaine, stovaine, durocaine, and organic compounds without number. We take novocain as a type. It may be applied:

1. Direct to mucous membranes.
2. Injected into groups of nerves.
3. Put in direct contact with nerve endings in the spinal canal. This, of course, is spinal anesthesia.

Spinal anesthesia depends upon the introduction of novocain or one of its compounds into the spinal space where it is rapidly absorbed or fixed by nerve roots, producing anesthesia and loss of motor function of these roots, whether voluntary or involuntary. Its action is governed by certain well-defined laws. First, with a given quantity, the range of anesthesia is governed by the amount of

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solvent fluid used. By rapidly injecting a concentrated amount we may limit anesthesia to the perineum only or by barbotage, in which the spinal fluid is withdrawn and the drug added to it in the syringe before reinjection, extend it to dangerous heights in the spinal canal. Second, the larger the quantity of anesthetic used by volume, the higher will be the level of anesthesia. Third, the specific gravity or sinking quality of the dilution used will allow us to govern the level of anesthesia by regulating the position of the patient. Some solutions such as Pitkin's spinocaine will rise and other solutions, such as scurocaine (French preparation of novocain) will sink. Fourth, the duration of anesthesia is in proportion to the concentration of the material used.

It will be seen that in the hands of a skilled operator this anesthetic may be fairly well controlled as to field and that it may be administered without physical shock. However, it is not consistent in its physiologic action, it can not be removed once it is administered and, as we shall see, it may produce a profound and disastrous effect which cannot be foretold.

It may definitely depress respiration. Too high an anesthesia may produce a respiratory paralysis, a frequent cause of death on the table. (Beecher[1]). It frequently produces a definite fall, sometimes profound, in the blood pressure, which may occur early and is usually proportional to the volume of injected fluid. This is due to paralysis of the vasomotor nerves. Secondary falls which are almost always serious are due to depressed vasomotor functions of the carotid sinus which may occur when the anesthesia reaches the level of the umbilicus. At this time the body loses its reflex capacity to compensate for hypotension, and hemorrhage or injury which the normal animal could easily withstand will produce circulatory collapse. (Cushney[2]). According to Maxon[3], this suggests that the only one hundred per cent safe spinal anesthetic is the saddle type which only anesthetizes the perineum and is ideal for prostatic resection or perineal operations. Any neurologic changes which may occur, either delayed or immediate, are usually more annoying than serious. (Davis, 1931).

As to the mortality rate we can get any figures we wish from the literature, from one death in 100 to one in 3,000. It would seem to me any anesthetic about which there is so much discussion as to its safety would be dangerous without further investigation. Note the lack of figures giving the mortality rate of ether. I would like to quote a statement of Bower[4], a co-worker of Babcock, from the *Cradle of Spinal Anesthesia*: "Spinal anesthesia is responsible for more deaths than any other anesthetic in proportion to the number administered . . . the mortality diminishes with the experience of the operator." Compare spinal anesthesia with our own old standby, ether. It is difficult to kill a patient with straight ether!

Let us now discuss general anesthetics. We will first consider those which are given by injection either intravenously or rectally. Here again we have anesthetics which cannot be stopped after having been started and which are inconsistent in their effects. We shall first consider avertin or tribromethanol. This has been widely used as a basal anesthetic, being a little too unsafe as a general. Its permanent place in the armamentarium of the anesthetist is questionable. Sebenning[5] considers it a highly complicated anesthetic which depends on its rate of absorption for its effect rather than the final quantity absorbed. This rate of absorption is so varied that it cannot be easily evaluated. Influencing factors are variations in tone and peristalsis of the bowel, and variations in its gas and fecal content. The "sleeping time" of this anesthetic is also difficult to gauge; it may be dangerously prolonged in diseases of the biliary tree, cachexia and sepsis. It may cause fatty degeneration of the liver; it is a marked depressant to the vasomotor center (Sollman[6]) and its administration is frequently followed by a serious decline in blood pressure. Some patients are peculiarly susceptible to this effect of the drug, and in them the extent of the fall is unpredictable. This would be an important reason for abandoning all but small (less than 100 mg.) doses in adults. The control of the administration is crude; it cannot be interrupted with any degree of surety and after twenty minutes cannot be removed from the colon. Rapid injection is dangerous; too slow injection leads to failure. It is contraindicated in liver and kidney diseases, cachexia, anemia,

sepsis, myxedema, ileus, partial obstruction of the bowel, tumors of the colon and colitis, where vital capacity is reduced, and in the aged. Hence it can be said it is only relatively safe in minimal doses as a basal anesthetic. It does not fulfil any qualifications of the ideal anesthesia except that it is easy on the patient and surgeon.

If a basal anesthetic to be administered rectally is desired, paraldehyde would be my personal choice. However, I do not feel that there are many reasons for giving a basal anesthetic when we consider we are starting an action which we cannot reverse. Giving a general anesthetic on top of a basal one is like driving a car with the throttle set at 25 miles an hour. We do not see many cows on the road but when one steps in front of the car we may want to stop in a hurry.

Next come the barbitals, as intravenous anesthetics. The chief ones are iso-amylethyl barbiturate, evipal and pentothal. The first has had its day, the latter two, particularly pentothal, are now very much in favor in some quarters. The action of all three is quite similar. Unfortunately, although many successful anesthesias have been carried out by intravenous doses of these compounds, this procedure is to be considered dangerous in that the dose taken, which is therefore beyond the control of the anesthetist, must necessarily approach the lethal one, resulting in a mortality rate which is greater than that of the volatile anesthetics. This anesthesia frequently shows late pulmonary complications resulting from prolonged respiratory depression. Postoperative mania and excitement may ensue. Most authorities hold that intravenous injection of the barbiturates should never be resorted to except in cases of emergency such as convulsions where rapid action is desired and where rectal administration is not favorable. (There is also the question of idiosyncrasy, which must not be ignored).

We will now consider anesthetics of the inhalant group. These are governed by the laws of absorption and exchange of gasses, which is a rapid and easily reversible reaction. Those of the ether group are characteristically fat soluble, and the amount of anesthetic required by a given patient is roughly in proportion to the amount of fatty tissue present, a valuable clinical fact.

Let us first consider ether. It is easily reversible, easily controlled, and obtains the best relaxation of any of the anesthesias, not excluding spinal which is not desirable in cases of ileus. However, it is difficult to anesthetize the patient rapidly, there is considerable psychic shock, and recovery is unpleasant. In these respects it fails to fulfil the requirement of the ideal anesthetic. Perhaps the great popularity and continued use of ether over a period of one hundred years is due to its safety. It may be considered to be the safest anesthetic. The mortality rate of ether is conservatively given by Cushney[2] as 1 death in 12,000 administrations.

Its ready controllability is due to the fact that its neurological signs are constant and consistent in almost everyone as the patient passes from one stage to another of anesthesia. It is so constant that the study of the various stages of ether anesthesia is usually taken as the standard for comparison with other anesthesias, and for teaching purposes.

The physiologic action of ether will be taken in detail. On the central nervous system its action is the same as that of alcohol. (This explains why persons who metabolize alcohol rapidly anesthetize poorly and suggests they should be prepared with 3 oz. of good whiskey). Its action starts with the highest cerebral centers, those of self-control, and passes down through the lower intracranial divisions to the medullary center, which is the last to be paralyzed. Wild movements in the early stages are caused by this loss of self-control, and the patient may be aroused by external stimuli such as noise and restraint. It is imperative that there be quiet in the operating room.

On the respiratory center ether produces a reflex stoppage in the early stages (which can be controlled by the newer methods of administering nitrous oxide, oxygen and ether), irritation of the center with gasping and irregularity in the second stage of excitement and dulled response in the third, or surgical stage. In the fourth stage there is increasing paralysis of the center which leads to respiratory failure. (Not cardiac failure). Note the ease of resuscitation.

With ether, as with any other anesthetic, the heart must carry more than its normal load of work, and with an impaired heart the signs of failure may be anticipated, but it is to be remembered that with ether the respiration always fails before the heart. The blood pressure with ether rises slightly in the early stages and then falls to normal.

Ether produces slight impairment of liver function which disappears the next day, in contrast to chloroform which produces permanent damage. Nitrous oxide produces no effect on the liver, unless accompanied by cyanosis, when both delayed and immediate toxic effects are observed. Evidently ether is the best anesthesia in case of diseased livers, as it is difficult to administer nitrous oxide without slight cyanosis.

In the blood ether increases the concentration of the red cells about 10 per cent. It produces a rapid rise in the white cell count, the most marked of all anesthetics. It generally triples the white count in about eight hours, taking four to five days to return to normal. The blood sugar shows a rise of from 100 to 200 per cent with ether. The coagulation time is decreased from 1 to 24 per cent due to the action of the drug on the adrenals. (Nitrous oxide on the other hand, has been shown to increase the coagulation time). Shock is not particularly great but is more pronounced with ether than with nitrous oxide.

At this point I may mention that in the administration of ether during tonsil operations, the cooperation of the surgeon is necessary in the first plane of the third stage to maintain an open airway. Closure of the airway by excess mucus will result in spasms and anoxemia which in children may produce disastrous results in the early part of the operation. Excess mucus in throat operations is frequently caused by crowding the anesthetic and producing too great a vapor tension in the first stage. It is essential that the patient should be in the second stage with a return of swallowing reflexes at the end of the operation before he is taken off the table. This will reduce the incidence of post-tonsillary lung abscesses and pneumonia.

To sum up, ether is safe but it is unpleasant to take and is uncomfortable to come out of.

Chloroform and ethylchloride are mentioned only to be condemned. The frequency of ventricular fibrillation and serious heart damage with small concentration of these anesthetics preclude their use and discussion.

Divinyl ether is an anesthetic of the alcohol-like group which is exceedingly rapid in its action and not unpleasant to take. Its administration must be accompanied by a large amount of oxygen. The rapidity with which it goes from one stage to another calls for care and skill in its administration. It is relatively safe in experienced hands but should be generally confined to induction and short operations. When it is used to induce ether anesthesia, great care should be used in switching over, that too great a combined anesthetic tension at this point does not produce disaster.

Ethylene will not be discussed. Apparently most clinics don't mind losing a patient once in a while, but when they lose the patient and the anesthetist and the surgeon get burned, it is a little too much and they stop using it.

Cyclopropane is a new anesthetic, rapid in its effects and easy on the patient. It is given in low concentration of from 6 to 20 per cent with oxygen. It has very little side effect except prolongation of bleeding time. Apparently its only disadvantage is that cardiac failure occurs before respiratory paralysis in the deep stages. Otherwise it is the ideal anesthetic.

Nitrous oxide is a gas which exerts a specific action on central nervous tissues, reinforced by anoxemia present. (Since 94 per cent nitrogen is necessary for complete anesthesia there is always a slight amount of anoxemia present). (Cushing, 1938). The circulation and heart are not affected by nitrous oxide except as to the amount of anoxemia present.

Nitrous oxide pushed to the final stages simply paralyzes the respiratory muscles, hence the treatment of accidents under nitrous oxide consists of artificial respiration alone. There is always considerable warning before respiratory failure so that even in the deep fourth stage there is a small margin of safety. This anesthetic when combined with small quantities of ether (20 to 40 cc. with the closed method) and used with carbon dioxide as a respiratory stimulant when needed is as near

the ideal anesthetic as we have. The induction is easy on the patient, who is under control at all times, and good relaxation can be obtained. The stages of anesthesia are well defined and as the ether may be almost entirely removed from the patient by the end of the operation, recovery is rapid, the average patient showing a swallowing reflex within two minutes after the mask is removed.

To administer nitrous oxide successfully, the preparation of the patient and method of induction are exceedingly important. There is no anesthetic in which different types of patients show such a varying response in the first and second, or excitement, stages. For purposes of discussion we shall divide people into the vagotonic individuals, in whom the bulbosacral nervous system is predominating, and the sympatheticotonic type which shows an overstimulation of sympathetic nervous system. The vagotonic is familiarly the heavy-set, pleasant individual who never worries and takes life easy. He has a tendency under anesthesia to show early and constant contraction of the pupils, bronchial and laryngeal spasm and intestinal hyperistalsis. He typically vomits and shows cyanosis and rigidity while in the deeper stages of anesthesia. The sympatheticotonic individuals will show dilated pupils through the second stage which may be so marked as to appear much deeper than they really are. With them it is almost impossible to tell what stage of anesthesia they are in, except by their respiration. They may struggle, vomit, gag and show excess mucus in the first stage in contrast to the quiet first stage of the former type. It is sometimes impossible to get them beyond the first plane of the third stage (or deep enough to operate upon) without the addition of a small quantity of ether.

I feel that patients may be further subdivided into the introverts and the extroverts. The former will be quiet in the first stage and require restraint in the second. The extrovert will as a rule take a rough and noisy first stage.

With nitrous oxide, the point of induction marks the beginning of the first stage. At this point there should be absolute quiet in and about the operating room. Remember that for five minutes in the first stage noises and conversation impress themselves upon the subconscious mind of the patient and are reflected as motor responses. The slightest movement of the patient's arms or legs in the first stage may provoke respiratory spasms and clonic motions. These involuntary spasms in the early stage should be differentiated from the purposeful struggling of the hysterical or ornery patient. This type of patient fights the anesthesia with purposeful movements. It may be due to fear, but is usually due to lack of self-control or a simple showing off. This contingency is usually best met by taking off the mask and giving the patient a severe talking to. In five minutes the patient enters the second stage of delirium and this is when the attendants must stand by to avoid any accident. In the late second stage muscle tone increases, the jaws set, and the eyes close, and the body stiffens. At this stage too rapid an induction or too high a vapor tension of anesthesia will produce respiratory paralysis.

To bring all our patients to as near a mean level as possible is the purpose of preliminary medication. From experience and observation I have found that everyone should have a grain and a half of nembutal the night before and the morning of operation. The vagotonic types should have a large dose of atropine, the sympathetic types need little or none. Small doses of morphine are of an advantage to the extroverts of both groups. The morphine should not be increased above one-sixth unless pain is present, when enough should be given to make the patient comfortable. Preliminary medication properly given to selected patients will make nitrous oxide, oxygen, ether anesthesia consistent in all cases.

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SCIENTIFIC PROGRAM — 1941

The Committee on Scientific Work through its chairman, Dr. Herbert E. White, has mailed to each member of the State Medical Association a letter outlining the routine to follow in making application for a place on the program of the Sixty-eighth Annual Meeting to be held in Jacksonville, April 28-30, 1941. Members desiring to read papers at the next annual convention should make application prior to January 6, 1941, as no paper will be accepted after that date.

The Committee desires papers that are worth while, interesting and well prepared, and that deal with the various specialized fields as they relate to the general practice of medicine. It is important that applications be forwarded as early as possible, for the task of reviewing carefully all papers submitted and selecting those that will make up a program of varied interest, arranging for proper representation from all parts of the state, will require a great deal of time and study. Members wishing to apply for a place on next year's scientific program are requested to follow closely the outline of procedure as indicated below.

1. Submit a paper on any subject in which you are particularly interested. (We are anxious to get papers that contain original thought or new ideas in medicine; possibly a paper read before your county medical society.)
2. Name two members whom you desire to serve as discussors.
3. No paper shall occupy more than 15 minutes in its time of delivery.
4. Mail your paper (preferably in full) or a 500-word synopsis of it to the chairman of the Committee on Scientific Work, P. O. Box 1018, Jacksonville, so that it will arrive not later than January 6, 1941. No paper will be accepted after that date.

MEDICAL PREPAREDNESS

The Army and Navy have requested the doctors of the nation through the American Medical Association to cooperate in selecting men for the new army who will be chosen from those who registered on October 16, 1940. There are other activities also in which we must be of service to the nation, both to the citizens at home and to the men in our defense forces. As the initial step in this work, a meeting was held in Chicago at the headquarters of the American Medical Association on September 20, 1940. Representatives of the Army, Navy, Public Health Services and various divisions of the American Medical Association were present. The medical needs of both the army and the civilian population and the means by which the doctors can help to meet these needs were discussed. Your state representative, Dr. Edward Jelks, was proud to report the progress which has been made in Florida.

Perhaps it would not be out of place for us to review at this time what has already been done. There has been appointed by the President of the United States, as the liaison between the Council of National Defense and military and organized medicine, a Committee which is composed of Dr. Irvin Abell, the three Surgeon-Generals and the Chairman of the National Research Council. Its immediate contact with organized medicine will be through the National Committee on Medical Preparedness of the American Medical Association, the personnel of which has been printed several times in the *Journal of the American Medical Association*. To serve under and function with this Committee, there has been appointed by each state president a state committee and these state commit-

tees in turn have formed local committees of the various component county medical societies. Thus, based upon the structure of organized medicine, there is now a very intimate connection between each doctor and his country's welfare—through the county committee, the state committee, the national committee, the liaison committee, and the Council of National Defense. It is the hope of every committee man that he may make some contribution in service to his country and to each doctor in organized medicine.

It is already known that physicians of the country will be called upon to help in the organization of the new army, for it is proposed that on each local draft board, of which there will be about 6,500 in the country, there will be one doctor whose duty it will be to examine registrants who have been selected by the board. There will be established also medical advisory boards to aid in settling questions of physical fitness in the doubtful cases. The personnel of both the local draft boards and the medical advisory boards are to be appointed by the President on the recommendation of the Governor. Your State Committee on Medical Preparedness has officially offered the assistance of organized medicine to the state authorities and has been assured that it will be called upon to help in the selection of the appointees to these boards.

The first service we can render to the Council of National Defense is to provide information about each individual doctor in the nation. In order to accumulate this information, the American Medical Association has sent a questionnaire to every doctor of medicine. Although the response to these has been very satisfactory in some sections, the general average is not good. The state reporting the highest returns was Nebraska with 88.3 per cent returns; the lowest reported only 40 per cent. Florida had a return of 64.1 per cent. After all reasonable efforts to secure these questionnaires have been exhausted, the Committee on Medical Preparedness of the American Medical Association must send to the military authorities some sort of report, and in regard to those who did not send in the requested information, it will have to rely upon the inadequate material contained in the Medical Directory of the American Medical Association. These incomplete data obviously will not be a satisfactory description of a doctor's ability and standing and will be unfair both to the Council of National Defense and to the doctor himself; so let us urge you to *fill out your questionnaire and send it in at once.*

Be assured that the members of your County Committee want to be of any service possible to the members of the county medical society. They will receive information from time to time as to the activities of the various committees on medical preparedness. Elsewhere in this Journal there is printed a list of the various committees in Florida.

DISTRICT MEETINGS "D," "E," AND "F"

All arrangements have been made for the second series of three medical district meetings. Dr. Robert B. McIver, chairman of the Council, has completed the programs for the scientific sessions. The local committees on arrangements for the entertaining societies have met with Dr. Stewart Thompson, managing director of the Association, and formulated plans for entertainment of the doctors, guests and ladies. Printed programs were mailed from the Association's home office to all the members of the three districts a week or ten days in advance of the meetings.

This will be the fourth annual meeting in each medical district. The attendance has increased steadily from year to year as the doctors realize that this is a splendid opportunity to meet their colleagues informally; to enjoy a program of good scientific papers, which this year includes a guest speaker from some other medical district in the state, and to become better acquainted with the officers of the State Association. Following are the places and dates of each of the three medical district meetings and the names of members of the local committees on arrangements.

DUNEDIN—October 31. Local committee on arrangements: Drs. J. A. Mease, Jr., chairman; H. E. Winchester and E. M. Harrison.

FORT PIERCE—November 1. Local committee on arrangements: Drs. H. D. Clark, chairman; R. C. Boothe and M. D. Council.

CORAL GABLES — November 2. Local committee on arrangements: Drs. Herman Boughton, M. M. Coplan, and Hillard W. Willis, co-chairmen.

MEDICAL MEETINGS ARE OPEN TO MILITARY DOCTORS

Many physicians in the United States military service are moving from one location to another in Florida. It is suggested that the various county medical societies in Florida contact such doctors, as the opportunity arises, and invite them to attend medical meetings. Unless they possess Florida licenses to practice medicine, they are, of course, not eligible for membership in the State Association. However, there are many medical questions of mutual interest and the officers of your Association feel that while a doctor in military service is in your locality he should be invited to attend your county medical society meetings and take part in the discussions, even though he would not have the rights of a voting member.

WARNINGS

Physicians in Alabama and Tennessee have complained that a Mr. C. W. Myers, who claims to be representing F. A. Davis Company of Philadelphia, has taken orders for books, collected for them at the time and failed to deliver them.

He will probably attempt to continue his fraudulent operations in the southern states. Any information should be referred to the Better Business Bureau in your city or Philadelphia, or to F. A. Davis Company, 1914 Cherry St., Philadelphia.

Mr. C. W. Myers no longer represents Paul B. Hoeber, Inc., Medical Book Department of Harper and Brothers, and at no time has he had authority to collect money for the purchase of Hoeber medical books. As he probably still has some sales material from this firm, he may still be offering their books. Any doctor receiving a visit from this person is requested to notify Paul B. Hoeber, Inc., 49 East 33rd Street, New York, by collect telegram.

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WASHINGTON-HOLMES—R. H. Segrest, Bonifay, Chairman; F. M. Watson, Chipley; George W. Carter, Chipley.

BIRTHS AND MARRIAGES

BIRTHS

Dr. and Mrs. W. L. Jennings of Daytona Beach announce the birth of a son, William Logan, Jr., on August 31, 1940.

* * *

Dr. and Mrs. Don C. Robertson of Orlando announce the birth of a daughter, Mary Ann, on July 21, 1940.

* * *

Dr. and Mrs. Young L. Hall, Jr., of Miami announce the birth of a son, Young Lafayette, III, on July 31.

MARRIAGES

Dr. W. M. Hoover, Jr., of Miami and Miss Amelia Wylie were married on August 15.

STATE NEWS ITEMS

Dr. Henry Fuller of Mulberry was the guest speaker at the local Kiwanis Club's weekly luncheon, Tuesday, August 13. Dr. Fuller's address covered the need for a full time county health unit, his arguments being based on mortality statistics.

* * *

Dr. C. C. Box of Graceville spent the latter part of August visiting clinics, and vacationing in the North Carolina mountains.

* * *

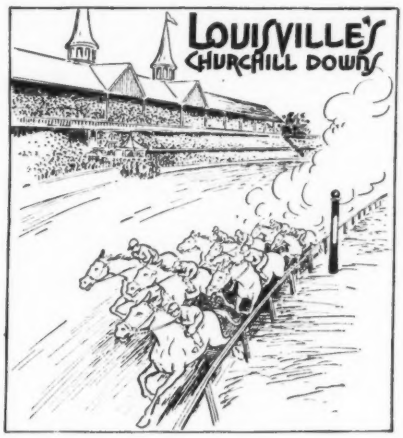
Dr. Meredith Mallory of Orlando was the principal speaker at the local Rotary Club meeting, August 12. Dr. Mallory's address was on the so-called socialized medicine.

* * *

Dr. Harrison G. Palmer of St. Petersburg returned in September from a five weeks vacation spent in North Carolina, Michigan and Ohio. He attended a number of clinics while away.

* * *

Dr. M. A. Lischkoff of Pensacola is enjoying his fall vacation in New York City.



AS "the Derby" determines the winner among equestrian thoroughbreds, so each meeting of the Southern Medical Association becomes more established as a consistent winner among the thoroughbred medical meetings. See another winner when the Southern Medical Association meets in Louisville, Kentucky, November 12-15, 1940.

MEDICALLY, there may be expected the usual fine programs and entertainment that distinguish the annual meetings of the Southern Medical Association from the others. Ten general clinical sessions, nineteen sections, the three independent medical societies meeting conjointly, and outstanding scientific and technical exhibits are assurance that every phase of medicine and surgery will be available.

REGARDLESS of what any physician may be interested in, regardless of how general or how limited his interest, there will be at Louisville a program to challenge that interest and make it worth-while for him to attend.

ALL members of state and county medical societies in the South are cordially invited to attend. And all members of state and county medical societies in the South can be and should be members of the Southern Medical Association. The annual dues of $4.00 include the Southern Medical Journal, a fine publication recognized as a valuable instrument to physicians of the South in the pursuit of their professional careers.

SOUTHERN MEDICAL ASSOCIATION
Empire Building
BIRMINGHAM, ALABAMA

Train schedules and railroad fares from various cities in Florida to Louisville, Ky., where the annual meeting of the Southern Medical Association will be held, appear on page 207 of this Journal. Note advertisement of the Atlantic Coast Line, Central of Georgia, and Louisville & Nashville Railroads.

* * *

Dr. George M. Green has started construction of a 10 bed hospital as an addition to his office in Daytona Beach. It will be of modern design, concrete construction. The building will be completed in about three months.

* * *

Dr. T. Hartley Davis of Shamrock announces the opening of offices for the practice of general medicine at Bradenton, October 1, 1940.

* * *

The Association's Committee on Legislation and Public Policy met at the Thomas Hotel, Gainesville, Sunday, September 29. This was the first official meeting of the Committee since the annual meeting in Tampa. Those present were Drs. J. Maxey Dell, chairman; W. C. McConnell, H. D. Van Schaick and B. M. Rhodes. Advisory members were Drs. J. Sam Turberville, president, and Stewart Thompson, managing director.

* * *

Dr. J. Maxey Dell, Jr., of Gainesville presented an exhibit at the meeting of the American Roentgen Ray Society, October 1 to 4, inclusive, at Boston. The exhibit was on the x-ray diagnosis and treatment of chronic sinus disease in infants and children. It consisted of 30 cases taken from a series of 85 cases illustrated by films taken before and after treatment; also a follow-up history of from nine months to two years, from Dr. Dell's experience.

* * *

The name of the late Dr. Joseph Y. Porter was honored recently by the Key West Housing Authority. The white project which will consist of 136 family units situated on the property now purchased on Trumbo Island, bounded by the extension of White Street beyond Eaton Street and the western boundary of the navy air field, will be known as Joseph Y. Porter Place. The late Dr. Joseph Y. Porter was state health officer for some thirty years, a member of the local county medical society, the State Medical Association and the American Medical Association. Dr. Porter died on March 16, 1927 at the age of 80 years.

* * *

Dr. John A. Simmons of Arcadia has spent a month's vacation in Waynesville, N. C., Niagara Falls and Buffalo, N. Y., Canada, Washington, Virginia and South Carolina.

* * *

Dr. E. J. Melville of St. Petersburg returned home recently after a four month stay at Melville's Landing on Lake Champlain.

* * *

Dr. Charles L. Park of Sanford shattered 10 out of 15 targets to win the first competitive shooting in Mo-skeet-o, a pygmy skeet range, at the barbecue given by the Orange County Medical Society, August 8.

* * *

Dr. W. L. Shackleford of West Palm Beach was a state delegate to the 42nd annual convention of the American Hospital Association, held in Boston, the middle of September.

COMPONENT COUNTY SOCIETIES

DADE

The regular monthly meeting of the Dade County Medical Society was held on Wednesday, September 4, at the Holsum Baking Company building. Dentists and druggists of Dade County were guests. The group was conducted through the plant after which a buffet supper was served by the bakery. The program which followed consisted of 2 papers:

"A Study of Peripheral Vascular Disease in the Negro" by Dr. S. Charles Werblow; discussed by Drs. E. Sterling Nichol and Max Dobrin.

"The Clinical Application of Some Newer Forms of Vitamin Therapy" by Dr. James L. Anderson; discussed by Drs. Paul Kells and William H. Izlar.

* * *

DUVAL

The October meeting of the Duval County Medical Society was held on the evening of the 1st in the Library of the State Board of Health. Dr. Robert M. Baker presented a paper on "New and Advanced Methods in the Treatment of Specific Urethritis" which was discussed by Drs. E. T. Sellers and E. W. Veal. A business meeting was then held after which refreshments were served.

JACKSON

The Jackson County Medical Society held its regular meeting on Tuesday, August 27. Following a dinner at the Hotel Chipola, a scientific program was presented:

"The Ethical Relationship of Doctor and Patient", Dr. J. Sam Turberville, Century. "The Medical and Surgical Management of Splenic Anemia", Dr. D. A. McKinnon, Jr., Mayo Clinic, Rochester.

"Carcinoma of the Cervix Complicating Pregnancy", Dr. R. N. Joyner, Marianna.

Several guests were present: Drs. R. D. Crawford, J. T. Ellis and S. G. Latiolais of Dothan, Ala.; Drs. W. G. Miles and C. K. Hayes of Chattahoochee.

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PASCO-HERNANDO-CITRUS

Dr. and Mrs. S. C. Harvard of Brooksville entertained the Medical Society at their home Thursday evening, September 12. A grilled steak dinner was served after which a scientific meeting was held.

The Pasco-Hernando-Citrus County Committee on Preparedness was appointed after discussion, said committee consisting of Drs. W. H. Walters, Lacoochee; S. C. Harvard, Brooksville; and George Dame, Inverness.

Dr. W. H. Walters spoke on the Civic Health Council of Pasco County and explained what was being accomplished by this organization. Reports on interesting cases were given and discussed.

Present were: members—Drs. C. L. Carter, G. R. Creekmore, S. C. Harvard, W. W. Jones, W. H. Walters; visitors—Drs. R. P. Fike and Bobby Hodges of Atlanta.

* * *

PINELLAS

Dr. C. C. Rudolph of St. Petersburg was principal speaker at the meeting of the Pinellas County Medical Society held on the evening of September 6. His subject was "Roseola Infantum alias Exanthem Subitum".

On the evening of September 20, members of the society, their ladies and friends gathered at the Penguin Club to honor the retiring president and his wife, Dr. and Mrs. John A. Herring. After a social hour, a steak dinner was served.

ABSTRACT DEPARTMENT

Members of the Florida Medical Association who have had articles published in out-of-state medical journals are requested to forward such journals or reprints to Box 1018, Jacksonville, for abstracting in this department.

Occupational Dermatitis Due to Mint, SAMS, WILEY MITCHELL, Miami, *Arch. Dermat. & Syph.* 41: 503-505 (March), 1940.

Two patients, both bartenders at Miami Beach, were found to have a contact dermatitis, involving mainly the fingers and hands. Patch tests, involving all materials used in mixed drinks, were performed and both were positive only to mint. Proscription of mint handling resulted in quick cessation of symptoms.

An interesting feature was that only in Florida were they annoyed with this dermatitis. This was ascribed to the fact that here "horse" mint or Mentha citrata was used while in the North, the common mint used for mixed drinks was Mentha vera which apparently has less eczematizing properties than the citrata.

Productive Aortitis with Multiple Aneurysms in a Child, NEIMAN, BENJAMIN H., Chicago, and MARKS, MEYER B., Miami, *Am. J. Dis. Children* 59: 571-578 (March), 1940.

The patient was first seen in March, 1935, at which time she presented pustules with necrotic centers over the face, buttocks, and tibia. The Mantoux test was positive 1-10,000. The Wassermann was negative. Diagnosis at this time was pulmonary tuberculosis with papulonecrotic tuberculids.

In November she began having attacks of dyspnea and slight elevation of temperature. Both the head and neck were swollen and full. The lips were swollen and cyanotic. The veins of the neck were engorged, and the eyes were prominent. The fingers and toes were cyanotic and showed definite clubbing. The radial, brachial and axillary pulse were absent. There was marked precordial pulsation and the cardiac dulness was widened transversely.

Roentgenological reexamination showed a mediastinal mass projecting into the upper fields of the left lung and increasing gradually in size in succeeding films. Death occurred nine months after the initial admission. Wassermann tests on the father and mother were negative.

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The essential findings at autopsy were multiple aneuryisms involving the thoracic aorta, one of which impinged on and partially compressed the left subclavian artery.

Microscopically, there was fibrotic thickening of the intima, degeneration and fibrosis of the media and diffuse sclerosing inflammation of the adventitia, all of which was characteristic of syphilitic aortitis in the acquired form of the disease. In the absence of serological confirmation and the negative family history, the authors were unable to conclude that this was definitely syphilitic in origin and discussed also the possibility of a rheumatic origin for the condition.

BOOKS RECEIVED

Acknowledgment of books received will be made in this column and this will be deemed by us a full compensation to those sending them. A selection will be made for review, as expedient.

NEW AND NONOFFICIAL REMEDIES, 1940, containing descriptions of the articles which stand accepted by the Council on Pharmacy and Chemistry of the American Medical Association on Jan. 1, 1940. Cloth. Price, postpaid, $1.50. Pp. 656-LXVIII. Chicago: American Medical Association, 1940.

Each year a revised list of the articles which stand accepted by the Council on Pharmacy and Chemistry of the American Medical Association as of January first is published in book form under the title of "New and Nonofficial Remedies." The book contains the descriptions of acceptable proprietary substances and their preparations, proprietary mixtures if they have originality or other important qualities, important nonproprietary nonofficial articles, simple pharmaceutical preparations, and other articles which require retention in the book.

A list of articles and brands accepted by the Council, but not described, is included in the book to cover simple preparation or mixtures of official articles (U. S. P. or N. F.) marketed under descriptive, nonproprietary names for which only established claims are made. Diagnostic reagents which are not used in or on the human body, and protein diagnostic preparations are not included in New and Nonofficial Remedies unless the determination of the status of these products by the Council has been requested by the distributor: If such products are found to be marketed in accordance with the Council's rules, they may be included in the list of undescribed, but acceptable articles.

New and Nonofficial Remedies is a practical and condensed text of pharmacology and therapeutics; it contains scientifically elaborated standards for all accepted nonofficial drugs; its Index to Distributors is a list of manufacturers, a large number of whose products have met the Council's high standards; its Bibliographical Index is a storehouse of references to reports which have been made mainly on unaccepted and unacceptable drugs; its prefatory material contains the Council's "Rules," a time-tested and reliable set of basic principles for the furtherance of scientific and rational medicine.

A supplement to the annual volume of New and Nonofficial Remedies is published twice a year to bring up to date such current revisions and additions as have been necessary since its last publication. Every product included in the book is subject to the official rules of the Council. The comments to rules are changed occasionally by way of clarifying interpretation to insure fair consideration of all submitted preparations as new standards are recognized. Such constant and critical consideration of its contents provides the physician with a valuable reference list of acceptable new preparations on which to base his selection for use in treatment according to the established current practices of the profession.

The 1940 New and Nonofficial Remedies, of course, contains the revisions which appeared in the supplements for the 1939 edition, and continues the plan of grouping together articles having similar composition or action under a general discussion. These discussions have undergone considerable revision in the 1940 edition. Further revision of statements regarding the actions, uses, dosage, composition, purity, identity, strength or physical properties of many of the articles has also been necessary in some cases. Noteworthy revisions are those of the chapter on Liver and Stomach Preparations, radically rewritten and including a statement of requirements suggested by findings of the Anti-Anemia Preparations Advisory Board of the U. S. Pharmacopeia; the subsection Tuberculins, entirely rewritten to conform to newer knowledge in this field; and the chapter Allergenic Protein Preparations, the name of which has been changed to Allergenic Preparations. Minor but relatively important revisions are found in the articles: Bismuth Compounds, Serums and Vaccines, and Vitamins and Vitamin Preparations for Prophylactic and Therapeutic Use.

The indices of the new volume of New and Nonofficial Remedies are of the same order and plan as in previous editions. A general index lists accepted articles, including those not described. This is followed by an index to distributors in which appear all the Council accepted articles listed under their respective manufacturers. Finally, a bibliographical index is added for listing proprietary and unofficial articles not included in N. N. R. This includes references to the Council publications concerning each such article as has appeared in The Journal of the A.M.A., Reports of the Council on Pharmacy and Chemistry, Propaganda for Reform, Vol. 1 and 2, or Reports of the A.M.A. Chemical Laboratory.

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THE UNSEEN PLAGUE: CHRONIC DISEASE. By ERNST P. BOAS, M. D.,Chairman of the Committee on Chronic Illness, Welfare Council of New York City; Assistant Clinical Professor of Medicine, Columbia University. This book consists of a review of the extent of chronic diseases in the community and their effect on the social structure; also a summary and appraisal of the present methods of medicinal and social care. It is based on twenty years of active participation in the field and is intended to serve as a basis for planning and action by government, community, physician and individual. Cloth, pp. 121, price $2.00. New York, J. J. Augustin, Inc. 1940.

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THE MARCH OF MEDICINE. New York Academy of Medicine, Lectures to the Laity, No. 4. For some years the New York Academy of Medicine has sponsored highly successful popular lectures on interesting phases of medical history as seen with the perspective of modern medicine. These lectures bring out the highlights of medical progress. Gathered together here in book form, those for 1938 and 1939 make an exceedingly entertaining volume. In content they range from the subject of health in Elizabethan England to the romance of modern surgery. And while this work is sufficiently authoritative to prove of value to medical men implemented and enriched with a knowledge of the history of their profession, it is sufficiently simple and clear to prove of special value to those who have marginal interest in medicine such as biology teachers, health educators, instructors in physical education, nurses, scientists, and technicians. Cloth, pp. 168, price $2.00. New York: Columbia University Press, 1940.